Cell, Vol. 120, 151–152, January 14, 2005, Copyright ©2005 by Elsevier Inc. DOI 10.1016/j.cell.2004.12.032

Erratum

A Behavioral Role for Dendritic Integration: HCN1 Channels Constrain Spatial Memory and Plasticity at Inputs to Dendrites of CA1 Pyramidal Neurons

It has come to our attention that panels D, E, and G in Figure 6 of Nolan et al. (Cell *119*, 719–732, November 2004) are incorrectly labeled. The symbols for control (*HCN1*$^{f/f}$) and forebrain-restricted *HCN1* knockout (*HCN1*$^{f/f,cre}$) are reversed. The closed symbols in these panels correspond to data from *HCN1*$^{f/f,cre}$ mice, whereas the open symbols refer to data from *HCN1*$^{f/f}$ mice. Note that throughout the rest of the paper, closed symbols refer to data from *HCN1*$^{f/f}$ mice and open symbols refer to data from *HCN1*$^{f/f,cre}$ mice. The correct figure is shown here.

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(Figure 6 is on the following page.)

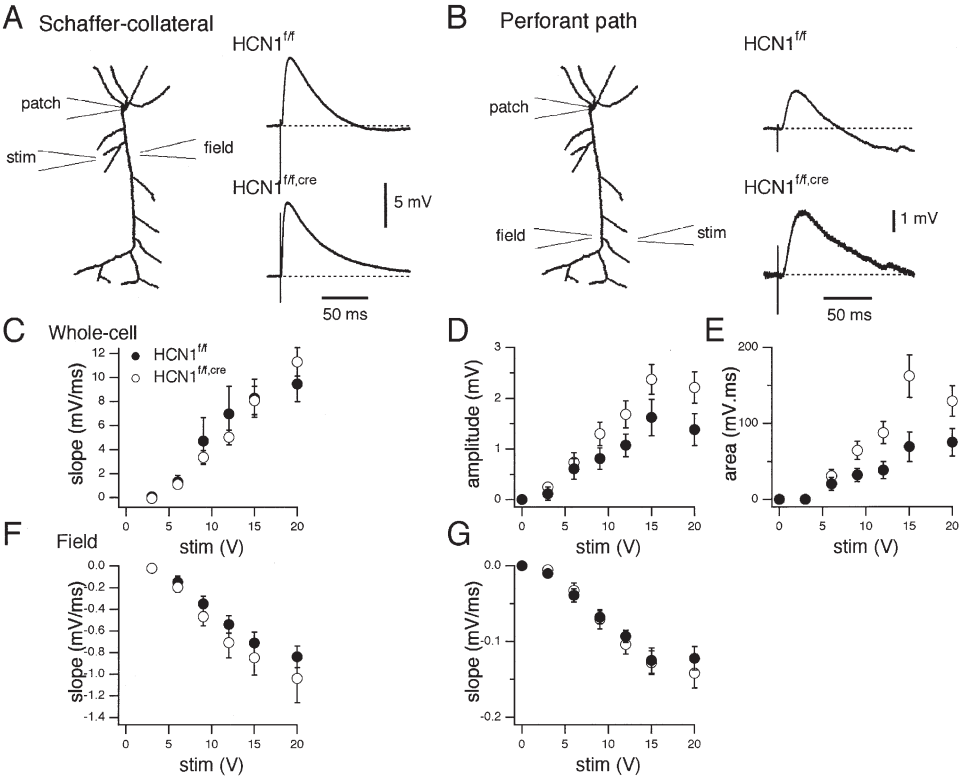

Figure 6. Effects of HCN1 Knockout on Schaffer Collateral and Perforant Path EPSPs in CA1 Pyramidal Cells

(A and B) Schematic illustration of the recording configuration and examples of somatically recorded EPSPs evoked by stimulation of Schaffer collateral inputs (A) and perforant path inputs (B). Subthreshold Schaffer collateral EPSPs from *HCN1*$^{f/f}$ mice (n = 8) and *HCN1*$^{f/f,cre}$ mice (n = 10) had 10%–90% rise times of 6.3 ± 0.9 ms and 6.7 ± 1.6 ms, respectively (p = 0.84), decay times of 38.6 ± 7.0 ms and 51.3 ± 4.8 ms (p = 0.14), and areas of 366.2 ± 55.9 mV.ms and 617.2 ± 74.6 mV.ms (p = 0.02). The mean amplitude for *HCN1*$^{f/f}$ and *HCN1*$^{f/f,cre}$ Schaffer collateral EPSPs respectively, was 10.6 ± 1.6 mV and 11.5 ± 1.7 mV (p = 0.7) with a stimulus of duration of 0.1 ms and intensity of 6 ± 0.8 V and 5.7 ± 0.5 V (p = 0.75). Subthreshold perforant path EPSPs from *HCN1*$^{f/f}$ mice (n = 8) and *HCN1*$^{f/f,cre}$ mice (n = 12) evoked by stimuli of amplitude 15 V and duration 0.1 ms had 10%–90% rise times of 7.14 ± 0.52 ms and 8.82 ± 0.80 ms, respectively (p = 0.14), halfwidths of 38.8 ± 6.4 ms and 57.6 ± 4.6 ms (p = 0.03). The mean amplitude for *HCN1*$^{f/f}$ and *HCN1*$^{f/f,cre}$ perforant path EPSPs, respectively, was 1.6 ± 0.4 mV and 2.4 ± 0.3 mV (p = 0.1) in response to stimuli with amplitude of 15 V and 1.7 ± 0.5 mV and 3.59 ± 0.6 mV (p = 0.04) in response to stimuli with amplitude of 25 V.

(C) Plot of the relationship between stimulus strength and the slope of EPSPs in CA1 pyramidal cells, from *HCN1*$^{f/f}$ mice (n = 6) and *HCN1*$^{f/f,cre}$ mice (n = 8), evoked by stimulation of Schaffer collateral inputs.

(D and E) Plot of the amplitude (D) and the area (E) as a function of stimulus strength for perforant path EPSPs recorded from CA1 pyramidal cells.

(F and G) Plot of relationship between stimulus strength and the slope of field EPSPs recorded from stratum radiatum (F) (*HCN1*$^{f/f}$, n = 10, *HCN1*$^{f/f,cre}$, n = 16) or stratum lacunosum moleculare (G) (*HCN1*$^{f/f}$, n = 8, *HCN1*$^{f/f,cre}$, n = 11) in response to activation of Schaffer collateral or perforant path inputs, respectively.